for the helpless and hopeless condition in which all the other members had been placed. After using ammonia, brandy, and digitalis, both subcutaneously and by the mouth, also heat and general faradization, without any apparent effect, unless it was to stimulate the patient's tongue, coffee was tried, a clear, large cupful being administered inside of two minutes. The effect was all that could be desired. The pulse soon improved, the pain and sense of oppression about the heart were relieved; in fact, all the disagreeable symptoms, except the stiffness of the muscles, passed away very rapidly, greatly to the relief of patient and physician.

POISONING BY ANTIFEBRIN.

The following case illustrates the dangers of self-medication. A healthy young married woman, who had been in the habit of taking antifebrin for headache, feeling pain come on one morning took, fasting, about a teaspoonful of the drug in some water. In about ten minutes, the headache not being relieved, she repeated the dose, which her husband remarked might prove dangerous. She consequently took a glass of milk and some alum water, in order to produce vomiting, which she succeeded in doing, but immediately afterward giddiness, singing in the ears, throbbing in the temples, and a dull pain in the head, together with a feeling of weakess, came on, and the face assumed a livid hue. About four hours after collapse occurred, and for three and a half hours her condition appeared hopeless. Among other things, the intravenous injection of a solution of common salt was used, and appeared to act most beneficially.

The patient was out of danger about fourteen hours after the drug had been taken.—*Lancet*, May 24, 1890.

MEDICINE.

UNDER THE CHARGE OF

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THE MORPHOLOGY AND DEVELOPMENT OF THE BLOOD.

ALEX. EDINGTON (*British Medical Journal*, 1890, i. 1233) gives an interesting account of the state of our knowledge regarding the blood, and details his own observations upon the subject. He refers to the very varying size of the red blood-cells, and the readiness with which this variation takes place. There is, namely, a distinct variation in the aggregate size between meals, the minimum occurring soon after a meal, while the maximum is seen at the end of a period of fasting. He had also observed that the cells are diminished in size at the termination of acute fever after an exanthem, while they are largest during the fever. They are small in septic conditions which have lasted some time. He takes the ground that the corpuscle is surrounded by an investing membrane, formed by a condensation of the stroma.

In discussing the white blood-cells he refers to the various forms, as determined by the difference in size, character of the protoplasm, number of the nuclei, etc. He describes at length the methods to be employed in examining the blood, recommending that various plans be followed in every case, in order to eliminate error as far as possible. Staining is often an essential, and for this purpose he prefers the aniline dyes, especially dahlia, methyl-violet, and methyl-blue.

His study of the morphology of the white cell has convinced him that there is an advancing process by nuclear division, until the uninucleated cell becomes one of four nuclei. The protoplasm meanwhile increases in amount, until the final four-nucleated stage is reached, when it begins to stain less readily, as if it were becoming dissipated. The division of the nuclei in the white cells is not an evidence of necrobiosis, as has been claimed, for the characteristic regularity of the process is sufficient evidence against this. Moreover, this phenomenon in other cells is followed by division of the cell itself, and in the case of the white blood-cell such division does not occur. Were the white corpuscles derived from the lymph glands, we could hardly find so many multinucleated forms as we do, since the lymph corpuscle is a cell which possesses one large nucleus. Observation has shown him that the correct view is that when the white blood-cell has reached the four-nuclear stage, the nuclei are set free by the protoplasm being used up or otherwise dispersed, and are then in a condition to act their parts as uninuclear cells. From this view it is evident that white corpuscles may be produced from pre-existing cells within the general circulation.

The author then takes up the various descriptions which have been given of the hæmatoblasts or blood-plates, in order that this body may not be confounded with what he denominates "a new blood corpuscle." These bodies, which he names "albocytes," he believes to be undoubtedly the young form of the red blood-cell. They are small, very delicate, spherical bodies, difficult to detect until the observer has accustomed himself to seeing them. They have nothing whatever to do with fibrin formation, and do not tend to dissolve as Hayem's hæmatoblasts are said to do. In a slide of fresh blood prepared with a sealed cover, they may even be seen after the lapse of a week. In the presence of fluids of specific gravity less than that of blood serum, they suffer from osmotic change just as do the red blood-cells. It is possible to see that the larger of them are distinctly discoid, and one can easily follow their transition up to the condition of the true red blood-cells. The author, therefore, claims that the red blood-cells are derived from these bodies by their gradual growth and change in shape, and by the requirement of hæmoglobin. He has been able to discover the origin of these small corpuscles after long search, and gives a reproduction of a photo-micrograph illustrating their morphology. This is, namely, as follows: The uninuclear white blood-cells possess a large nucleus which stains intensely, and which is surrounded by a narrow ring of protoplasm, which, after a short time, assumes a very ragged appearance. The same method of staining will, however, reveal certain other white blood-cells, whose unusually large nucleus stains but faintly. Within this nucleus is a nucleolus, also staining faintly; and within this again a very small endo-nucleolus, which is very darkly stained. Within the nucleus, and surrounding the nucleolus, may be seen a number of ill-defined small spherical bodies, the young albocytes, which become liberated in the blood under conditions not yet known to us. The white blood-cells containing these may be looked upon as mother cells, and he therefore denominates them "matricytes." The ring of protoplasm surrounding the nucleus in these cells differs from that in the ordinary white blood-cell in the length of time during which it remains intact. The albocytes may be distinguished from the granular matter of the blood by the fact that the granules possess a peculiar greenish, refractive appearance.

Finally, in discussing the granular matter of the blood, the author divides it into two sorts, one of which, probably the hæmatoblasts of Hayem, is quite evidently associated with fibrin formation, and which tends to break down in the way Hayem describes. The other sort of granules, which do not break down, might easily be mistaken for monads or bacteria. This kind he has observed to be extruded from the white blood-cells while they were undergoing amœboid movements. On being liberated they perform gyratory movements, and dance away across the field. They may also be noticed in an active Brownan movement. He believes that both of them are of the same origin, namely, from the white blood-cells; so that the white corpuscles are the chief agents in inducing fibrin formation, and of these corpuscles the granules of the first sort are the essential cause. The second variety of granules are of a similar nature, but older and undergoing degenerative changes, and have, therefore, lost their power of inducing fibrin formation.

PERNICIOUS ANÆMIA.

NEUSSER (*La Sem. Méd.*, No. 79, 1890) divides pernicious anæmia into two forms—the primary and the secondary. Primary pernicious anæmia is a clinical entity opposed to chlorosis, leucæmia, and pseudo-leucæmia. Secondary pernicious anæmia may be provoked by many diseases, as by frequent hemorrhages; neoplasms; osteo-sarcomata; carcinomata, especially those of the stomach and the spleen; ulcers of the stomach and intestine; chronic nephritis; infectious diseases (tuberculosis, syphilis, diphtheria, septicæmia, malaria); diseases of the blood, as leucæmia and certain forms of myelogenic pseudo-leucæmia; intestinal parasites, as *bothriocephalus latus, tænia mediocanellata, ankylostomum duodenale*, and even *oxyuris vermicularis*. In the parasitic form, however, the diminution in the number of the red blood-cells rarely reaches that seen in the primary forms. The anæmia usually disappears with the expulsion of the parasite, unless the organism has been already too seriously affected. There may be the coincidence of primary pernicious anæmia with the presence of a tænia. As regards the pathogeny of primary pernicious anæmia, several factors favor its development, such as unhealthy external conditions, defective nutrition, psychic depression, and especially the puerperal state and lactation. In the anæmia of pregnancy the red blood-cells are considerably diminished in number, and the leucocytes equal 5000 to 6000 per cubic millimetre. If, however, the anæmia of the pregnant or parturient woman is due to a septic condition, leucocytosis may be observed, which sharply distinguishes it from the primary, nonseptic variety. Except when it follows a severe hemorrhage or occurs during pregnancy, pernicious anæmia begins during health. The symptoms are those characteristic of grave anæmia, and are described by the author. The gastric symptoms witnessed may be due to atrophy of the gastric tubules, or to simple impoverishment of the blood, or to a degeneration of the posterior columns of the cord, such as has been seen in some cases. They would then be of the nature of *crises gastrique*. The liver and spleen are usually increased in size. The fever seen in anæmia seems to be in direct connection with the diminution in the number of red blood-cells. The toxic substances derived from the hæmoglobin may exert a pyrogenic action, exciting the centre regulating heat; or perhaps the hæmoglobin or the products of its decomposition destroy the leucocytes and the hæmatoblasts and produce an intoxication of the organism by setting at liberty the fibrin ferment. This auto-intoxication may explain other symptoms, as the occurrence of embolism and thrombosis of the small vessels and some of the nervous troubles. The coincidence of febrile reaction and of embolisms in pernicious anæmia may give rise to errors in diagnosis. Thus there are cases of ulcerative endocarditis in which the clinical appearance is identical with that of pernicious anæmia. In the former, however, there are an irregularity of the heart and an alteration of the second sound at the apex.

The number of red blood-cells in pernicious anæmia may be diminished even to 500,000; the number of leucocytes diminishes in the same ratio, but the hæmoglobin, though also reduced in quantity, is not so in proportion to the loss in red cells. The presence of leucocytosis indicates a secondary anæmia. The appearance of nucleated red blood-cells may have considerable prognostic value. So long as they do not surpass in size that of the normal red blood-cell, and so long as the nucleus stains easily with the ordinary reagents, it may be concluded that the regeneration of the blood is going on in the normal manner.

PERNICIOUS ANÆMIA AND SARCOMATA.

ISRAEL and **LEYDEN** (*Berlin. klin. Wochenschr.*, 1890, 231) discussed before the Berlin Medical Society a case whose history was much as follows: The patient, a woman of about thirty years, exhibited an enlargement of the spleen, then disappearance of the patellar reflex, anæmia, irregular fever, diarrhœa, and severe pains in various parts of the body. Later, there were ascites and bilateral pleural exudate. Numerous small tumors then appeared in the skin. The blood exhibited a decided leucocytosis, and, beside this, the character of pernicious anæmia, being thin, and with abundant microcytes. Further, the large nucleated red blood-cells, which Ehrlich considers characteristic of pernicious anæmia, were present in this case in numbers which Leyden had never seen before. It seemed reasonable, then, to classify this case as one of pernicious anæmia; and yet the matter was not certain, on account of the enlargement of the spleen and the large number of leucocytes present, as well as of the fact that the appearance of the patient was not that of a typical case of pernicious anæmia.

The autopsy showed some fatty degeneration of the heart muscle, but by no means to the extent seen in pernicious anæmia. The spleen was about ten inches in length, with abundant pulp and enlarged follicles. There were found tumors of various sizes, but none larger than a cherry, and though often widely disseminated through the muscles, they clearly took their rise from the external surface of the periosteum of the bones.

Nodular tumors were present in the marrow of the bones, chiefly in the long bones; the marrow was red and lymphoid in situations where there were none of the yellowish tumors. A few other nodules were found in other parts of the body; namely, on the pleura and peritoneum, and in the thyroid gland, the uterus, and pancreas. The macroscopical characters were those of the growths seen in leukæmia, but the microscopical appearance was quite different. The tumors were a large-celled growth, the cells being round, or often possessing processes, or spindle-shaped, or still oftener being very similar to those seen in the bone-marrow, though there were no giant-cells. Israel cannot answer the question as to the relation existing between the pathological conditions of the different systems of the body, and calls attention to the rarity of such numerous periosteal sarcomata. Leyden says that the autopsy revealed a combination of a sort of leukæmia, pernicious anæmia, and multiple sarcomata, but does not know in what way they stand related to each other.

ON THE EXCRETION OF HÆMATIN AND OF IRON IN CHLOROSIS.

HOESSLIN (*Munch. med. Wochenschr.*, 1890, 248) is of the opinion that there is no reason to separate chlorosis from other forms of anæmia as a peculiar constitutional disease. Virchow's opinion, that it is due to a congenital narrowness of the arterial system, is incorrect, as is shown by the fact, since learned, that the vessels which he considered abnormally narrow were not in reality so. The author is also opposed to the view that there is present in the disease any characteristic condition of the blood, such as a diminution of the amount of hæmoglobin without proportionate reduction in the number of red blood-cells; claiming that this relation may exist in other varieties of anæmia as well.

It is a very frequent occurrence that a girl, previously entirely well, becomes in the course of a few weeks extremely anæmic and chlorotic. Examination of the blood shows a great diminution in the amount of hæmoglobin; and the question arises as to how this loss has taken place. The author believes that it is not due to destruction of the blood within the vessels, and inclines to the view that it is simply the result of hemorrhage, and especially of concealed gastric hemorrhages. In the effort to substantiate this theory he examined the stomach in a large number of autopsies in different diseases, and found, he believes, the evidence that this, of all the organs, is peculiarly liable to suffer from hemorrhages of greater or less degree. He then made over 200 estimations of the amount of iron and of hæmatin present in the stools of normal persons and of chlorotic individuals, and found that in the latter both the iron and the hæmatin were decidedly increased.

That women, rather than men, are attacked by chlorosis is due, first, to the smaller amount of iron in their nourishment; second, to the smaller amount of HCl contained in their gastric secretion, on account of which a sufficient amount of iron is not dissolved and absorbed; third, to the losses of blood sustained in menstruation.

Other points which the author advances in proof of his theory are: 1. The beneficial results following the administration of iron are due partly to the fact that the chloride of iron formed in the stomach from the iron administered is one of the best known hæmostatics; thereby checking the loss of hæmoglobin at the same time that it increases its formation. 2. The fact that cardialgia and digestive disturbances so usually attend chlorosis. 3. The greenish-yellow color of the skin, hitherto unexplained, is probably due to the resorption of hæmatin and hæmoglobin from the intestine, and their transformation into other coloring matters. 4. The fact that the disease usually does not appear before the time of puberty, and disappears after the climacteric.

WARNINGS OF GENERAL PARALYSIS OF THE INSANE.

SAVAGE (*Brit. Med. Journ.*, 1890, i. 777) was for several years in the habit of getting friends of patients with this disease to fill in a printed sheet relative to the affection. In this way he obtained nearly a hundred such, and from these, combined with experience in consulting practice, he has drawn a considerable number of facts which he believes to be trustworthy.

General paralysis is, he believes, a degeneration rather than a specific disease, and is most commonly seen in middle-aged married men, inhabitants of cities, flesh-eaters, and drinkers of alcohol. It frequently follows constitutional syphilis; and is not uncommonly related to head injury or to causes of nerve-tissue disease, such as those produced by lead.

There are two special forms of onset: the gradual and the sudden. In the former there is a steady and progressive degradation of mind and body, so that the highest faculties show the first evidence of change. In the latter there is nothing to warn until suddenly a convulsive seizure, or an attack of emotional excitement or one of mania appears. Probably in both there were earlier changes going on which were hidden.

In many cases one meets with an account of early fatigue. This may precede the common symptoms by a year or more, and may for a time be replaced by a morbid buoyancy. This sense of fatigue is an important symptom, and is associated with indecision, doubt, gloominess, and even hypochondriacal weakness. It is only really valuable as a symptom when associated with predisposing conditions in which paralysis is common, and when one or more physical symptoms, as inequality of the pupils, or loss of power of expression by speech or writing, can be detected.

Ataxia may be the first symptom of general paralysis, or it may be a symptom of the disease occurring at any period during its progress. The author has known ataxic symptoms to be fully developed for several years before there was any suspicion of general paralysis. In these cases he has always found a history of syphilis. If the ataxy is part of general paralysis there will generally be muscular defect of the hands and tongue out of relation to the progress of the ataxy, and the symptoms will be either those of exaltation or of hypochondriacal melancholia; whereas, if the insanity be that of ordinary ataxy it will be of the suspicious or persecuted type.

One of the most striking and not uncommon of warning symptoms is temporary aphasia. This is not necessarily followed by general paralysis, but the author has seen cases in which this temporary aphasia has come on without apparent cause, and from this date there has been some defect of speech, the lips have been tremulous, or the expression faulty. It is common for these attacks of aphasia to recur at irregular intervals; and after several recurrences—it may be at intervals of a year or more—some other signs of disorder appear. In nearly all these cases disorders of speech and writing are early and clearly evident. The author has known such aphasic attacks occur nine years before symptoms of general paralysis developed.

Nearly related to aphasia, though usually coming after it, is a change in the handwriting. Some patients give up writing or alter their method of holding the pen for a year or more before signs of general paralysis are declared.

Facial expression is very early affected; the wiping out of the lines giving an appearance of fatness. Ptosis and external strabismus are rare as symptoms of general paralysis, but are very common as early symptoms of a diseased process which ends in general paralysis. Thus the author emphasizes his experience, that if after a history of syphilitic cranial nerve lesion there are any signs of nervous instability, there is reason to fear that general paralysis may be the result. In the same class of cases are the instances of serious head injury followed by local paralysis, which has passed off only to be followed by the steady mental and physical degradation which terminated as ordinary general paralysis. Recurring momentary slight or partial losses of power or sensation, sometimes comparable to attacks of *petit mal*, are warnings of general paralysis. An onset of fits of an epileptic type occurring in a middle-aged man, especially if he has had syphilis, prompt one to look for other signs of degeneration. If such fits occur at irregular intervals in an otherwise healthy middle-aged man, and if each fit is attended by marked mental changes, and if expression is in any way permanently affected, general paralysis is all but certain. These fits may precede the ordinary symptoms by several years.

Early sensory defects in general paralysis are not so easily recognized as are the motor alterations. Neuralgia, headache, sciatica, rheumatic pains, and the like antedated by a year or more the recognition of the disease in almost all the author's cases. Headache or facial neuralgia was the most common, in contradistinction to the rarity of headache in the insane. Double sciatica, especially if recurrent and associated with any change in character or habits, is a warning not to be overlooked. Temporary loss of sight, and alterations of hearing, taste, and smell, are sometimes early warning symptoms. Progressive loss of the highest refinement of some of the senses is manifest. Thus the general paralytic artist early loses the fine balance of tone and his colors become bold and crude.

In speaking of the intellectual changes which occur as warnings of general paralysis, the author calls attention to the loss of power of social accommodation as one of the earliest and most marked. The patient falls out of step with his fellows, though entirely unconscious of it. The actor forgets his part, the artisan makes misfits, judgment fails. Memory for recent events or engagements is defective, and this is especially marked if fainting or other fits have occurred. Loss of power of attention, want of persistence, and restlessness are marked. Moral weaknesses are common; such as stupid stealing of useless articles, and indecent exposure due either to lust or to negligence.

The next group of early symptoms may be provisionally classed as instability. This is evidenced as tremors of the finer muscles, uncertainty of gait, tendency to falls. It is a very common thing to hear that a fall has been the cause of the paralysis, whereas it was but the evidence of early paresis. This instability is marked almost always by the extreme readiness with which the patient is affected by stimulants, or drugs, or poisons. This evidence of weakness of the damaged brain is practically of great value.

Change of temper and character are probably the most constant of all the changes of early general paralysis. Change of character, instability of purpose, occurring with some motor weakness, almost always point to general paralysis if met with in a middle-aged man. Hypochondriasis may be one of the warnings of the disease. In such cases the morbid ideas are, as a rule, centred in the gastro-intestinal tract, and in addition there are physical signs, such as inequality of the pupils or speech defect, to aid us. The occurrence of marked hysterical or hystero-epileptic fits is even more alarming than the epileptic fits in middle-aged men, to which reference has already been made. The sudden outbreak of mania, especially of acute delirious mania, unpreceded by melancholia, and without apparent cause, is a frequent precursor of general paralysis.

PATHOLOGICAL ANATOMY OF FRIEDREICH'S ATAXIA.

BLOCQ and MARINESCU (*La Sem. Méd.*, 1890, 76) report the result of the examination of the cord from a case of Friedreich's ataxia. The cord was irregularly and considerably atrophied both in its transverse and its antero-posterior diameter. The columns of Goll were diseased throughout; those of Burdach irregularly involved as far as the decussation of the pyramids. The crossed pyramidal tracts were altered throughout their entirety, but the lesion decreased from below upward as far as the decussation. The cerebellar columns were attacked from the lower dorsal region; the columns of Gowers were not affected, at least in the limits which have been assigned to them. The portion of the gray matter corresponding to the zone of Lissauer was involved in the inferior lumbar region, but completely free above; the external marginal zone was unaffected; the columns of Clarke were profoundly diseased in all their extent; both fine fibres and cells.

They come to the following conclusions:

1. The spinal cord presented in this case a diminution in volume greater than has been observed by other authors.
2. The distribution of the lesions was almost the same as has been established for the disease in other cases. It differed, however, in that the zone of Lissauer was invaded, at least in the middle and lower regions.
3. Friedreich's ataxia is distinguished from tabes by its topography (the involvement of certain parts of the lateral tracts) and by the nature of the lesions.
4. It differs from certain cases of combined tabes in that the sclerosis is

more systemic, and is subject to the laws of degeneration, not extending by continuity.

5. In regard to the nature of the disease, it is the result of an hereditary predisposition transmitted to the spinal cord, and which materializes itself primarily by a vascular alteration. Dependent on the circulatory alteration there follows an atrophy of the organ, and, at the same time, a sclerosis.

The disease, in short, is a disorder of evolution, characterized by a special sclerosis occupying systemically certain determinate regions of the spinal cord.

THE CAPILLARY PULSE AND THE CENTRIPETAL VENOUS PULSE.

QUINCKE (*Berlin. klin. Wochenschr.*, 1890, 265) formerly searched for the capillary pulse in the finger-nail, but found this method often unsatisfactory, on account of the lack of transparency. He therefore rubs the forehead with some hard, smooth body, as the lower end of the stethoscope, until a red spot is produced by the paralytic dilatation of the capillaries and smallest arteries. In this the systolic increase of the size of the redness can often be well observed. For the production of the pulse there should be the greatest possible difference between the arterial pressure during the cardiac systole and that during the cardiac diastole. The capillary pulse is best seen in aortic insufficiency, and depends on the amount of blood regurgitated as well as on the energy of the succeeding ventricular contraction. To permit of much aortic regurgitation, not only must there be extensive valvular insufficiency, but the ventricle must be able to contract; for if it be dilated and weakened, and unable to expel more than a portion of its contents, there is evidently not much room for regurgitation. Consequently, in disturbances of compensation in cases of aortic insufficiency, the characteristic peculiarity of the pulse often disappears; and it is only with return of ventricular strength that capillary pulse and the celerity of the arterial pulse can be discovered.

The capillary pulse is not observed in every case of aortic insufficiency, and conversely it may be witnessed in other conditions also, wherever the *pulsus celer* is found, as in anæmic and chlorotic conditions, and in many nervous and muscular alterations of the cardiac action. It may sometimes be seen in the everted under lip, or on pressing a plate of glass against the finger tip. In fact, a certain slight degree of external diastolic pressure favors the recognition of the phenomenon. It may even appear in the natural redness of the cheeks, and in any localized area of inflammation of the skin. The other conditions being present, it is especially well seen in erysipelas.

Pulsation of the retinal arteries is developed under conditions exactly similar to, though more complicated than those producing the capillary pulse.

Different from the conditions for the production of the capillary pulse are the circumstances under which the pulse-wave extends from the arteries into the veins, and the centripetal venous pulse is formed. The general opinion is that it is of rare occurrence, but the author disputes this, and says that he has seen it dozens of times. The chief conditions requisite for its occurrence are relaxation of the vessels, chiefly the arteries, but also of the capillaries and veins. This is especially common in certain stages of fever, as in sudden fall of temperature, accompanied by profuse sweat. Thus he has seen it in a number of cases of typhoid fever, as also in recurrent and intermittent fevers, pyæmia, polyarthritis rheumatica, pneumonia, phthisis, and cholelithiasis. Nervous influences may join with the fever in producing it in such conditions as meningitis, spondylitis, encephalomalacia, and injuries of the cervical cord. He has also seen the venous pulse in afebrile conditions, as chlorosis and anæmia, and even in healthy persons whose peripheral vessels have been relaxed by the summer heat. It is witnessed in the veins of the forearm and back of the hand. Only once has he seen it on the dorsum of the foot. Besides the relaxation of the vessels, a number of other conditions seem necessary for the production of the phenomenon; such as thinness of the skin, a sufficiently powerful action of the heart, a certain degree of fulness of the veins dependent on the relaxation of the vessel-walls and the position of the arm at the time of examination. A slight change in the position of the member may be sufficient to cause the pulse to disappear. One must not expect to find the capillary pulse in every case of centripetal venous pulse; on the contrary, their combination is exceptional.

Though the later stages of fever are favorable to the production of the venous pulse, the condition which develops it to the best advantage is the combination of aortic insufficiency with relaxation of the vessels. Arteriosclerosis, also, may favor its occurrence, by transmitting a powerful pulse-wave into the smallest arteries. Venous congestion has not been, in his experience, liable to produce it.

SURGERY.

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PULSATILE EXOPHTHALMOS.

LEFORT (*Revue de Chirurgie*, June 10, 1890), in an exhaustive article upon pulsatile exophthalmos, to which are appended a bibliography and a complete list of cases, summarizes the subject as follows:

Pulsatile exophthalmos is an affection characterized by prominence of the eye and pulsations perceptible on palpating either the eyeball or the orbit. These pulsations are isochronous with the arterial beat, are accompanied by a bruit de souffle perceptible over a rather extensive area, and by intra-cranial bruits which are perceived by the patient.

The affection may originate in an aneurism of the ophthalmic artery or of the internal carotid, or in a phlebitis of the cavernous sinus, but is most frequently caused by a rupture of the carotid artery into the cavernous sinus. The abnormal development of the ophthalmic vein, to which the pulsations of the carotid are transmitted, is the principal lesion, in the sense that it is this which causes the principal symptoms.